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09/807,501	05/23/2001	Robert P. Kimberly	UAB-14402/22	9412

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EXAMINER

SITTON, JEHANNE SOUAYA

ART UNIT PAPER NUMBER

1634

DATE MAILED: 08/25/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/807,501

Applicant(s)

KIMBERLY, ROBERT P.

Examiner

Jehanne Souaya Sitton

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 6/1/2004.
- 2a) ☒ This action is FINAL. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-19,21 and 24-32 is/are pending in the application.
- 4a) Of the above claim(s) 4,6,8-10,12-19,21 and 24-32 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-3,5,7 and 11 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)  
Paper No(s)/Mail Date \_\_\_\_\_.
- 4) ☒ Interview Summary (PTO-413)  
Paper No(s)/Mail Date. \_\_\_\_\_.
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: \_\_\_\_\_.

### **DETAILED ACTION**

1. Currently, claims 1-19, 21, and 24-32 are pending in the instant application. Claims 1-3, 5, 7, and 11 are under examination at this time. Claims 4, 6, 8-10, 12-19, 21, and 24-32 are withdrawn from consideration as being drawn to non elected inventions. All the amendments, arguments, and declaration have been thoroughly reviewed but are deemed insufficient to place this application in condition for allowance. The following rejections are either newly applied, as necessitated by amendment or are reiterated. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow. This action is FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

3. The rejection of claim 20 under 35 USC 101 made in the previous office action, is withdrawn in view of the cancellation of the claim.

4. The rejections made under 35 USC 112/2<sup>nd</sup> paragraph are withdrawn in view of the amendments to the claims.

### ***New Grounds of Rejection and Objection***

#### ***Claim Objections***

5. Claim 2 is objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is

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required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form. It is unclear how claim 2 further limits independent claim 1.

***Claim Rejections - 35 USC § 112***

6. Claims 1-3, 5, 7, and 11 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue (See *In re Wands*, 858 F. 2d 731, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). These factors include, but are not limited to:

*Quantity of Experimentation Necessary*  
*Amount of Direction and Guidance*  
*Presence and Absence of Working Examples*  
*Nature of the Invention*  
*Level of predictability and unpredictability in the art*

Nature of the Invention

The claims are broadly drawn to determining susceptibility to any autoimmune disease or any cancer by haplotyping a Fas ligand promoter “region”, wherein the region is defined as from nucleotide -1032 to +33 wherein a polymorphism, T, at position -844

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is an indication of susceptibility to autoimmune disease or cancer. The claims are further drawn to the polymorphism being one that is active in binding NF-IL6 transcription factor.

Amount of Direction and Guidance

The specification teaches several genotype frequencies with regard to 4 polymorphic positions in the Fas ligand promoter in population studies of Caucasian and African Americans. The specification does not teach any specific haplotypes that indicate increased susceptibility to any autoimmune disease, any cancer, any specific autoimmune disease or any specific cancer. The specification does not teach any specific haplotypes that contain polymorphisms that bind NF-IL6 transcription factor. While the specification teaches a luciferase assay with regard to a T or a C allele at position -844 and that the C allele showed almost twice the activity than the T allele, the specification does not teach how such is correlated to binding of NF-IL6 or how any other allele would also be associated with binding such that the skilled artisan would be able to predictably determine alleles in the Fas ligand promoter 'region' which would be active in binding NF-IL6.

In addition, the claims are drawn to haplotyping in a 'region' wherein the specification does not define the metes and bounds of said region. Although the specification teaches and incorporates by reference, Holtz-Heppelmann et al (JBC, vol. 273, pages 4416-4423; 1998; see page 40 of specification) as teaching the region set forth in the claim, Holtz-Heppelman does not actually teach the full region set forth in the

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claim. Nucleotides 4-33 are not taught. Therefore, the claimed 'region' is not taught by the specification.

Further, the claims are drawn to determining any autoimmune disease or cancer by simply haplotyping the Fas ligand promoter and determining the presence of a T at an arbitrary position -844. However, the specification has set forth no predictable correlation that a SNP or particular haplotype is associated with increased or decreased susceptibility to autoimmune diseases or cancer in general. The terms "autoimmune disease" and "cancer" encompass an extremely large number of diseases and disorders, which have very different symptoms, causes (if known), and course of illness.

Autoimmune diseases encompass such diseases as SLE (systemic lupus erythematosus) and rheumatoid arthritis, for example, while cancers encompass leukemias, hepatocellular cancers, colorectal cancers, lung cancers, breast cancer, prostate cancer, etc. However, the specification provides no teaching or demonstration that patients with any specific haplotype or SNP would be susceptible to *any* autoimmune disease or cancer. The specification makes the general statement that the Fas ligand was found to be expressed in human melanoma, hepatocellular carcinoma, lung cancer, astrocytoma, esophageal carcinoma, and various other cancer, such teaching is not an indication that Fas ligand is necessarily involved in cancer, because many genes and proteins are expressed in cancer, without being directly or indirectly involved in such. In addition, although the specification teaches that certain alleles at position -844 occur with different frequencies in SLE vs RA, these frequencies are very different from each other and provide no predictable correlation that a specific allele or haplotype can generally be extrapolated to be associated with any autoimmune disease or any cancer.

Level of predictability and unpredictability in the art

There is a large body of knowledge in the prior art related to polymorphisms in general, and their association with diseases or disease states. However, the art is highly unpredictable with regard to the functionality of polymorphic sites in genomic DNA. After a screening assay identifies polymorphisms, it is unpredictable whether any such polymorphisms would be associated with any phenotypic trait, such as a disease state or a physiological state. For example, Hacker et al. teaches that they were unable to confirm an association between a gene polymorphism and ulcerative colitis in a case where prior studies suggested such a relationship would exist since the relationship had been identified in a different population (Hacker et al; Gut, 1997, Vol. 40, pages 623-627). Even in cases where an association between a particular gene and a disease state is known to exist, such as with the LPL gene and heart disease risk or the p-globin gene and sickle cell anemia, researchers have found that when using SNP (single nucleotide polymorphism analysis) it was difficult to associate SNPs with disease states or to even identify key genes as being associated with disease (Pennisi, Science, 281 (5384):1787-1789). Further, in some cases where multiple polymorphisms were identified in a gene, some of these were demonstrated to be disease associated and some were not. For example, Blumenfeld et al. (WO 99/52942) disclose a number of polymorphisms in the FLAP gene. While Blumenfeld et al. were able to demonstrate that some of these polymorphisms are associated with patients having asthma Blumenfeld et al found that some of these polymorphisms are not (see Figure 3). For example, the marker 10-35/390 was demonstrated to be associated with asthma, with a p value of 0.00229, while the marker 10-33/327 was determined not to have a statistical association with asthma

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( $p=0.294$ ). Thus, the art teaches that even for SNPs within the same gene, it is highly unpredictable as to whether a particular marker will be disease associated.

#### Presence and Absence of Working Examples

In the instant case, the polymorphism at position -844, and its incidence in different haplotypes was analyzed in different population samples. Statistically significant differences were found with regard to the occurrence of different genotypes in Caucasian vs African American populations, however such analysis does not predictably establish a correlation between a specific genotype and any autoimmune disease or cancer in general, or even any specific autoimmune disease or cancer. In addition, from the recitation in the tables it is clear that very different frequencies of haplotypes and genotypes occurs in two different autoimmune diseases. For example, a C at position -844 occurs in 63% of normal controls in the CAAC haplotype. As compared to these controls, only 44% of SLE patients possess the same allele, while 73% of RA (rheumatoid arthritis, an autoimmune disease) patients possess this allele. On the other hand in the TATC haplotype, a T allele at -844 is found in 12% of controls and only 5% of SLE patients but in 17% of RA patients. From this data it is completely unpredictable as to whether the occurrence of any particular haplotype or genotype increases or decreases an individual's susceptibility to any autoimmune disease.

With regard to the -844 allele, the specification teaches (page 35) that the C allele had twice the promoter activity than the T allele in a luciferase reporter assay. It is unclear, however, how such activity relates to binding of C/EBPB (NF-IL6). The specification further states that the low affinity allele (-844T) was over represented in



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patients with SLE (lines 15-18). The significance of this statement is unclear, however, because the teachings of Table 5 show that in one haplotype, TAAC, 42% of SLE patients had the T allele while only 24% of controls had the allele, but that in a different haplotype, TATC, the T allele was *underrepresented* in that 5% of SLE patients had the “low affinity allele” while 12% of controls had the allele. Therefore, it appears that the teachings of table 5 contradict the statement in the specification as to overrepresentation of the T allele in SLE patients. Further, if the term “overrepresentation” was with regard to 44% of SLE patients having the C allele and 56% of SLE patients having the T allele in table 5, without a teaching of the statistical significance of such data, the skilled artisan would be unable to establish predictable correlation with regard to the occurrence of a T at position -844 or any specific haplotype and susceptibility to autoimmune diseases or cancer in general or with any specific autoimmune disease or cancer.

The specification provides no analysis of a polymorphism at position -844 in the Fas ligand promoter in any patients vs controls with any type of cancer.

#### Quantity of Experimentation Necessary

Therefore, due to the lack of guidance from and unpredictability taught in the specification and the unpredictability taught in the art, undue experimentation would be required of the skilled artisan to practice the claimed invention. To practice the invention as claimed, the skilled artisan would be required to perform an analysis of the required position within the Fas ligand promoter in different types of autoimmune diseases and cancer, to determine whether any association exists between such polymorphisms in diseased patients as well as controls. Given the conflicting evidence presented by the

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specification between a polymorphism at -844 (T) in SLE vs RA patients (both are autoimmune diseases), such analysis would require an extremely large amount of unpredictable trial and error analysis. As both the specification and the art demonstrate that the outcome of such analysis is highly unpredictable, such experimentation is considered undue.

### *Response to Arguments*

7. The response traverses the rejection and presents arguments and a declaration under 35 USC 1.132. The amendments, response and declaration by Robert P. Kimberly have been thoroughly reviewed. The declaration was found persuasive with regard to the use of the term “putative” as well as the statistical significance of the data in table 1 showing that -844T was found in 56% and -844C in 44% of SLE patients. The remaining amendments, arguments, and portion of the declaration were found unpersuasive for the following reasons.

The response asserts that the claims are not directed to a predictable correlation between polymorphisms and any disease state or physiological state. This argument has been thoroughly reviewed but was found unpersuasive. It appears that the response mischaracterizes the previous office action, as the previous office action did not assert or state that the claims were drawn to a predictable correlation between any disease state or a physiological state. Rather, the previous office action set forth under “Nature of the Invention” that “The claims are broadly drawn to determining susceptibility to any autoimmune disease or any cancer by haplotyping a Fas ligand promoter...” The language drawn to ‘susceptibility’ was in fact acknowledged by the previous office

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action. The response further asserts that given the multifactorial nature of immune diseases, one of skill in the art would recognize that a change in FasL expression, while conferring susceptibility to immune disorder, may also manifest in various ways such that a particular phenotype may or may not be apparent and that while a particular polymorphism may be not be diagnostic for disease, it same polymorphism is diagnostic for susceptibility to disease. This argument has been thoroughly reviewed but was found unpersuasive. While such statement may be true in certain situations, in the instant specification, conflicting evidence is provided as to an association between the presence of a T at position -844 of Fas ligand and susceptibility to rheumatoid arthritis vs SLE. The occurrence of a C vs T at position -844 of Fas ligand was opposite in arthritis and SLE patients vs controls. Given such evidence, it is clear that the specification does not support that a generalized predictable correlation can be made between the polymorphisms as position -844 and susceptibility to any autoimmune disease or cancer, but reflects that further unpredictable trial and error analysis must be undertaken to determine whether such an assertion (susceptibility) can be made.

The response further asserts that the prior art shows that Fas and FasL mutations lead to autoimmune disease in mice and cites R. Watanabe-Fukunaga et al (Nature, 1992, vol 356, pages 314-317) and Takahashi et al (Cell, 1994, vol. 76, pages 969-976). This argument as well as the references cited have been thoroughly reviewed but were not found persuasive. Both Fukunaga et al and Takahashi et al refer to a "systemic lupus erythematosus like autoimmune disease" (see abstract of Fukunaga et al and page 969, col 2, first full para of Takahashi). Neither reference teaches that any mutation in Fas or FasL confers general susceptibility to all autoimmune diseases or cancers. The response

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further asserts that the specification teaches that the polymorphism at position -844 affects promoter activity of FasL. This argument has been thoroughly reviewed but was found unpersuasive as the specification does not teach how this polymorphism's affect on promoter activity confers susceptibility to the large number of autoimmune diseases and cancers encompassed by the broad scope of the claim. While the specification and the art teach that FasL is involved in apoptosis, neither the specification nor the art teach a predictable association between susceptibility to the large number of autoimmune diseases and cancers encompassed by the broad scope of the claim and any mutation or a specific mutation in FasL that affects the promoter activity.

The response further asserts that one of skill in the art would recognize that correlation of the identity of a particular FasL promoter polymorphism with SLE is indicative of a correlation with susceptibility to autoimmune diseases generally. The response asserts that the specification teaches at page 6, that SLE is considered the prototypic systemic autoimmune disease and that one of skill in the art would recognize that correlation of SLE with a particular susceptibility factor is indicative of a susceptibility factor for autoimmune disorders generally, including disorders such as systemic vasculitis, autoimmune lymphoproliferative syndrome, glomerulonephritides, Sjogren's syndrome, and IgA nephropathy. The response also asserts that the examiner must be taken as true by the examiner. These arguments, as well as the declaration, have been thoroughly reviewed but were found unpersuasive. Firstly, the examiner has not questioned that the specification states that SLE is considered 'a prototypic autoimmune disease'. However, the specification does not state or assert that SLE is representative of all autoimmune diseases, or cancer, whereas the claims encompass an association with a

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large number of different autoimmune diseases and cancer. Additionally, the specification has not taught what diseases are considered to fall within the category of “systemic autoimmune diseases”, such that one of skill in the art would be capable of determining which other diseases, SLE was prototypic for. It is noted that the declaration asserts that SLE is a prototypic autoimmune disease and provides corroboratory references for such assertion. A thorough review of the references, however, reveals that SLE is actually distinguished from other types of autoimmune diseases. For example, Vyse and Kotzin (Genetic Susceptibility to Systemic Lupus Erythematosus, *Ann Rev. Immunol.* 1998, pages 261-292) teach that while SLE is considered to be the prototypic autoimmune disease, but also teach that “unlike specific autoimmune diseases such as MS and type 1 diabetes mellitus, SLE has the potential to involve multiple organ systems directly...”. Such statement, as well as the references cited by the declaration, fail to provide evidence that mutations in FasL which are associated with susceptibility to SLE or diagnostic for SLE, are also associated with susceptibility to autoimmune diseases generally.

The response asserts that one of skill in the art would recognize, given the teachings in the specification, that a polymorphism in the FasL promoter is a factor in susceptibility of an individual to cancer. The response asserts that FasL is known to be present in numerous types of cancers and lists a number of different cancers. Further, the response asserts that the specification is not limited to the observation that FasL is expressed in different types of cancers, but that also “Recent evidence suggests that tumor infiltrating lymphocytes are susceptible to Fas-mediated counterattack” and that as such, the prior art at the time of filing recognized that “in esophageal cancer, the extent of

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apoptosis of TILs was found to vary regionally within the tumors in relation to the local status of FasL expression, local expression of FasL by nests of tumor cells was associated with apoptotic depletion of TILs". These arguments as well as those further presented with regard to colon cancers typically expressing FasL, as well as arguments directed to liver metastasis, have been thoroughly reviewed but were found unpersuasive. Particularly, it is noted that the response's assertions are directed to expression of FasL, that is, that FasL is expressed in different types of cancers and regions of tumors, whereas the specification has shown that the presence of a T at position -844 of the FasL is associated with decreased promoter activity (see page 35 of the specification). While it appears from the teachings in the prior art that FasL may be involved or associated in some way with TIL apoptosis, it is unclear how a mutation which decreases the promoter activity, and thereby presumably decreases expression of FasL, would be associated with an increase in susceptibility to cancer. The arguments in the response appear to conflict with the evidence of promoter activity associated with the polymorphism at position -844 of the FasL promoter. In summary, while the teachings of the specification provides evidence that the Fas/FasL system may be associated in some way with cancer, the specification provides no teaching or working examples that, or how, the polymorphism at position -844 (T) is associated with increased susceptibility to cancer in general.

The arguments at page 10, 2<sup>nd</sup> full para are persuasive with regard to an association between a T at position -844 in the FasL gene and increased susceptibility to SLE. It is noted, however, with regard to positions -756, -478, and -205, that association is found with specific haplotypes. Further, the examiner has not proposed claims for

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allowance as it appears that the specification fails to provide support for the sequence of FasL from -1032 to +33, as outlined above and in rejections below.

For these reasons, the rejection is maintained.

### *Indefinite*

8. Claims 1-3, 5, 7, and 11 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 1 is indefinite in the recitation of "Fas ligand promoter region extending from nucleotide -1032 to nucleotide +33, wherein a T at position -844..." because the nucleotide positions set forth in the claim are arbitrary absent a sequence for comparison. This rejection could be overcome by providing a SEQ ID NO: for reference, however it is noted that a SEQ ID NO: directed to positions -1032 to +33 would introduce New Matter into the claims because the reference of Holtz-Heppelmann, which the response uses for support for teaching the region (see page 40 of the specification), does not teach the full region but only -1032 to +3. Further, the specification does not provide any basis for a sequence from -1032 to +3 of Fas ligand.

Claim 5 is indefinite because it is unclear if the claim is drawn to any of the polymorphisms listed, in which case, as -844 was elected, it is unclear how the claim further limits claim 1, or if the claim is meant to indicate that haplotyping in claim 1 further encompasses haplotyping at positions -756, -478 and -205.

Claim 11 is indefinite because it is unclear which claim it depends from, as claim 8 is drawn to a non-elected invention.

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***Conclusion***

9. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the date of this final action.

10. No claims are presently in condition for allowance.

11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Benzion, can be reached on (571) 272-0782. The fax phone number for this Group is (703) 872-9306.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.



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Jehanne Sitton  
Primary Examiner  
Art Unit 1634

8/23/04